

GLYCIN AND MUSCULAR FATIGUE

What is potentially an epoch-making discovery in nutritional physiology was made about four years ago by Boothby¹ and Wilder² of the Mayo Clinic, who found that a daily supplementary feeding, with relatively large doses (15 grams) of glycine, increases muscular strength and skill, and delays the onset of fatigue, as determined by subjective symptoms. Moreover, in their hands supplementary glycine feeding tended to restore wasted muscle tissues in myasthenia gravis and related diseases. This observation is currently confirmed and placed on an objective basis by Ray, Johnson, and Taylor³ of the Department of Physiology, Long Island College of Medicine.

Since many individuals are unable to take the suggested large doses of glycine without discomfort, the Brooklyn biochemists have given their supplementary amino-acid feeding with foods rich in glycine—commercial gelatin, which contains about 25 per cent glycine, being selected for most of their tests. They found that as much as 30 grams of gelatin can be given in a single dose if suspended in eight ounces of well-chilled orange juice. The mixture, however, must be taken promptly before the gelatin swells appreciably.

In preparation for supplementary amino-acid feeding, six men and four women were maintained for a month on routine diets, and tested daily on an electrically recording bicycle ergometer. The maximal output of work before fatigue sets in was thus determined for each individual during the control month. The ten subjects were then placed on a supplementary gelatin diet, the daily supplementary intake being 60 grams of commercial gelatin for the men (or the equivalent of 15 grams of glycine), and about 50 grams of gelatin for the women.

Under this forced glycine feeding the maximum work output before onset of fatigue increased rapidly in the men, reaching its maximum in about thirty days. This maximum was usually at least twice the amount of work on the pregelatin diet. In one case the maximum was 340 per cent of the initial work output. In contrast with this beneficial effect in men, no appreciable increase in work output was recorded for the women. At the end of about six weeks, the supplementary gelatin diet was discontinued; the men, however, continuing to take the usual supplementary daily ration of orange juice. In all cases the work output before onset of fatigue fell rapidly, and was nearly as low as in the pregelatin period by the end of three weeks.

A theory as to the mechanism of this favorable action of supplementary glycine feeding on musculature efficiency was suggested by earlier investigators. Tripoli and Beard,⁴ for example, demonstrated a creatinogenic action of glycine, and a storage of at least part of the resultant creatine in

the muscles. Creatine storage in male muscle has been a recognized phenomenon for over a decade.⁵ There is no evidence, however, of appreciable creatine storage in the female. Failure of the four women to respond to the supplementary gelatin diet, therefore, is in line with this previously recognized sex difference.

Whether or not the beneficial effects of supplementary glycine feeding are confined to the skeletal muscles has not yet been determined. If future investigation should show the beneficial effects are shared by the cardiac muscle, or by certain smooth muscle structures, supplementary glycine feeding may in time become of wide clinical interest. Of particular interest would be a study of its effect on immunity mechanisms. A more palatable form of supplementary glycine feeding than that used by the Brooklyn physiologists should not be beyond commercial ingenuity.

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⁵ Rose, W. C., Ellis, R. H., and Helming, O. C.: *J. Biol. Chem.*, 77:171, 1928.

Diphtheria.—Just forty years ago diphtheria was so prevalent, so feared, and so contagious that it did not seem an unwarranted procedure for people to cross the street in a wide arc to avoid passing directly in front of the house quarantined with that disease.

Until scientific discoveries revealed the cause and the methods for the cure and control of diphtheria, few diseases had presented such high mortality, or before whose onsets mankind was so helpless. An outbreak of diphtheria in a community caused a shudder of horror. The old records are full of instances where all the children of a family were swept away in spite of what medical knowledge of the time could do.

Thanks, however, to modern researches, there are not few diseases about which so much is known. Its prevention and control are feasible, according to the United States Public Health Service (see "Diphtheria, Its Prevention and Control," Supplement No. 156, Public Health Reports), provided the intelligent coöperation of the sanitary authorities, the medical profession, and the general public is assured.

In the original registration states—Maine, New Hampshire, Vermont, Massachusetts, Rhode Island, Connecticut, New York, New Jersey, Michigan, Indiana, and the District of Columbia—the diphtheria death rate in 1900 was 40.4 per 100,000 total population. By 1910 it had dropped to 22.5; a decade later, to 17.3. About that time a still more rapid decline began, and the rate in the same group of states was 4.3 in 1930 and 1.3 in 1934. It fell from 3.9, in 1933, the first year such a figure was made, to 2.4 in 1936, for the total continental United States.

"Scientific medicine," according to the United States Public Health Service, "achieved one of its greatest triumphs when it placed in our hands the specific treatment for diphtheria—diphtheria antitoxin. Were it possible to apply this remedy in sufficient dose and early enough in all cases, the mortality from diphtheria would almost vanish. As it is, the disease has been robbed of much of its former terror."

Self-Protected.—Few physicians die of tuberculosis despite the fact that they are constantly exposed to it. Knowledge defends them as it may yet defend other groups in the population when properly educated in self-protection.

¹ Boothby, W. M.: *Proc. Mayo Clinic, Staff Meeting*, 9:600, 1934.

² Wilder, R. M.: *Ibid.*, 9:608, 1934.

³ Ray, G. B., Johnson, J. R., and Taylor, M. M.: *Proc. Soc. Exper. Biol. and Med.*, 40:157 (Feb.), 1939.

⁴ Tripoli, C. J., and Beard, H. H.: *Southern Med. J.*, 31:662, 1938.